Autonomic dysreflexia: guidelines for practice

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INTRODUCTION

Until the turn of the 20th century, a spinal cord injury (SCI) was considered to be fatal. In the First World War, about 90% of spinally injured people died. By the 1960s, about a third of all tetraplegics still did not survive (Spinal Injuries Association 1998). Today, the management of the spinally injured patient, involving a multidisciplinary and integrated approach, is greatly improved. The causes of early death, such as bladder and kidney infections, can be prevented and more patients with spinal injuries are surviving (Spinal Injuries Association, 1998).

Annually, there are about 10–15 new SCIs per million of the population in the UK, which approximates to about 750–1000 new patients per year (Swain and Grundy, 1996; Spinal Injuries Association, 1998). In the UK, it is impossible to find out the exact incidence of patients with spinal injuries because cases of SCI do not have to be notified to the Department of Health and there is no central register of these injuries (Spinal Injuries Association, 1998). Hambly and Martin (1998) state there are about 40,000 spinal cord injured patients in the UK, most of which have been caused by a physical injury, ranging from road traffic accidents to sporting accidents (Hambly and Martin, 1998).

Although patients with SCI are mainly looked after in special units, many will be admitted to an intensive care unit (ICU) at some point in time. Such patients may be admitted to an ICU for emergency care and stabilisation, following the onset of complications, such as respiratory failure, or following surgery, such as spinal fixation.

Spinally injured patients can present many challenges for nursing care: physical, psychological and social. Although most general ICUs do not admit significant numbers of spinal patients, it is important that nurses are fully aware of their needs and potential complications. Certain complications related to SCI may lead to acute situations that can be seriously damaging to the health status of paralysed individuals and may lead to death. Autonomic dysreflexia, also known as hyperreflexia, is one of these acute medical emergencies.

What is autonomic dysreflexia?

Some patients with injuries to the spinal cord above a certain level experience a disruption to the autonomic homeostasis afforded by the autonomic nervous system (ANS) (Pasquina *et al.*, 1998). Known as autonomic dysreflexia, it can occur with both incomplete and complete spinal cord lesions. It can present in mild or severe forms, with varying symptoms (Cormarr and Eltorai, 1995). It has been described as a paroxysmal reflex sympathetic discharge, in response to noxious (harmful) stimuli below the level of the cord injury (Colachis, 1992). If this dysreflexic cycle continues without intervention, serious consequences, such as seizures, cerebral haemorrhage, stroke and death may occur (LaFavor and Ang, 1997).

Autonomic dysreflexia is a serious and potentially life-threatening condition for patients with SCI. It can occur in 85% of tetraplegic patients (Vaidyanathan and Krishnan, 1996). It may be a troublesome complication, occurring soon after spinal shock, or it may not appear for the first time until many years after the original injury (Cormarr and Eltorai, 1995).

Prompt recognition and treatment

Immediate recognition and prompt and effective treatment of autonomic dysreflexia are of the utmost importance for all those involved in the care of spinal patients. Nurses who work with spinally injured patients on a regular basis are usually familiar with the onset of symptoms and management strategies. However, the pathogenesis and specific treatment measures are not widely known among nurses and carers who infrequently come across such patients (LaFavor and Ang, 1997). To avoid haemorrhage or death due to the paroxysmal hypertension of autonomic dysreflexia, prompt and early intervention is vital, and 'acquiring an understanding of how the ANS is affected by high thoracic and cervical SCI is necessary in order to appreciate how this dysfunction may lead to autonomic dysreflexia' (LaFavor and Ang, 1997, p.83).

The purpose of this paper is to focus on the specific problem of autonomic dysreflexia. It will discuss the relevant anatomy, physiology and pathothysiology; recognition of signs and symptoms; and preventative and emergency intervention. It aims to provide general ICU nurses with a clinical guide to support their care of spinally injured patients.

ALTERED PHYSIOLOGY IN SPINAL CORD INJURY

The spinal cord is the largest nerve in the body. Trauma to the head, neck, shoulders or back, resulting from a variety of causes, may cause injury to the vertebral column and/or the spinal cord (Hanak and Scott, 1993).

It is not always possible to correlate bony column injuries with spinal cord injuries. Usually in severe disruption of the vertebral column severe neurological damage results, but not always (Burke and Murray, 1975). Minor column disruptions may, or may not, be accompanied by neurological dysfunction. The spinal cord may be damaged by compression, without injury to the column, as in a hyperextension injury (Burke and Murray, 1975). In addition, viruses, cysts and tumours, on or near the spinal cord, can cause permanent damage leading to paralysis (Spinal Injuries Association, 1998). The spinal cord does not have to be completely severed to cause loss of functioning in the body.

Loss of movement is indicated by the word *paralysis* and muscle weakness by the word *paresis*. A more serious form of paresis is termed *plegia*, which is Greek for a blow or paralysis. Spinal cord injury is termed *paraplegia* when motor and sensory loss affects both legs and *tetraplegia* (or *quadriplegia*) when both legs as well as both arms are affected (Porth, 1998).

In individuals with high thoracic and cervical spinal cord injuries, clinical signs of autonomic dysfunction may be apparent. This is because the injury separates the parasympathetic branch of the ANS from the sympathetic branch, thus affecting the negative feedback loop. Parasympathetic fibres exiting the brain stem continue to stimulate the effector organs, as before the injury, but the sympathetic outflow is severely diminished. Stimulation of an intact parasympathetic system in the presence of a diminished sympathetic system can result in symptoms of autonomic dysfunction (LaFavor and Ang, 1997).

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is the result of the body's inability to restore autonomic equilibrium when presented with a noxious stimulus from below the level of the spinal cord lesion. It is a potentially life-threatening condition, typically encountered in spinal-injured patients, with lesions at or above T6. It has been reported to occur in 30–90% of such patients (Pasquina *et al*., 1998; Vaidyanathan and Krishnan, 1996).

Autonomic dysreflexia is an acute episode of exaggerated sympathetic reflex responses that occurs in people with SCI. It is usually triggered by noxious stimuli, occurring below the level of the SCI, that normally cause pain in the abdominopelvic region (Porth, 1998). Often the noxious stimulus arises within the bladder or bowel, in conditions such as distension, obstruction, impaction, infection, and digital stimulation (Grundy and Swain, 1996; Pasquina *et al*., 1998). Most commonly, it occurs due to visceral distension caused by a full bladder or a full bowel.

Stimulation of pain receptors, secondary to an irritating pressure sore or an ingrown toenail, and visceral contractions, such as ejaculation, bladder spasms or uterine contractions, may also trigger the syndrome (Burke and Murray, 1975; Porth, 1998).

Medical interventions, such as cystoscopy and lithotripsy, and in rare cases sexual intercourse (Vaidyanathan and Krishnan, 1996) and childbirth (Grundy and Swain, 1996) may precipitate autonomic dysreflexia. In fact, stimuli of any type below the level of the SCI can set off pathological reflexes, via somatic or autonomic nerve pathways, leading to autonomic dysreflexia (Cormarr and Eltorai, 1995).

SIGNS AND SYMPTOMS OF AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is characterised by an abrupt onset of excessively high blood pressure caused by uncontrolled sympathetic nervous system discharge. True autonomic dysreflexia is potentially life-threatening and is considered to be a medical emergency (Spinal Injuries Association, 1998). Due to the severity of the consequences of untreated autonomic dysreflexia, it is important that carers of SCI patients are well-informed about the syndrome. Rapid intervention can be life-saving.

Many of the signs and symptoms are secondary to hypertension. Other symptoms are secondary to the vasodilatory reaction of the parasympathetic nervous system. An individual with a SCI above T6 often has a normal systolic blood pressure in the range of 90–110 mmHg. A blood pressure of 20–40 mmHg above baseline may be a sign of autonomic dysreflexia (Consortium for Spinal Cord Medicine, 1997).

Table 1 lists the most common symptoms, which may be minimal. In addition, an individual may not experience them all at once (Consortium for Spinal Cord Medicine, 1997). Also, Pasquina *et al*. (1998) report symptoms of parasthesia, with LaFavor and Ang (1997), noting symptoms of chills without fever, changes in vision, and bronchospasm. Symptoms of nausea, lethargy, dyspnoea, choking, dizziness, tinnitus (Cormarr and Eltorai, 1995) and atrial fibrillation (Pine *et al*., 1991) may also be experienced.

PATHOPHYSIOLOGY OF AUTONOMIC DYSREFLEXIA

The autonomic dysreflexia response is mediated through the nervous system, which consists of the central nervous system (CNS) and the peripheral nervous system (PNS). The PNS is divided into the somatic nervous system (SNS) and the autonomic nervous system (ANS).

The ANS is involved in regulating, adjusting and coordinating visceral functions, such as blood pressure and blood flow, body temperature, metabolism and respiration. It is responsible for the ability to maintain homoeostasis via the parasympathetic and the sympathetic nervous system. The sympathetic division evokes a maximum energy response (known as the 'fight or flight' response). The parasympathetic division is primarily responsible for energy-conserving functions. The two branches balance each other by providing antagonistic innervation to most effector organs of the body (LaFavor and Ang, 1997).

In general, people with injuries at the T6 level or below have sufficient sympathetic outflow to control visceral reflexes. In people with SCI at T6 or above, the sympathetic responses that occur at and below the level of the spinal cord are lost. However, the baroreceptor function and parasympathetic control of the heart rate remain intact (Porth, 1998).

Impulses from noxious stimuli travel from the receptor (site

Table 1. The most common symptoms of autonomic dysreflexia*

Symptoms
There may be a pounding headache, secondary to hypertension and vasodilatation
A flushed face, red blotches of the skin and profuse sweating above the level of the lesion, and nasal congestion are all signs that may indicate autonomic dysreflexia. These are all secondary to vasodilatation
Piloerection and cold and clammy skin below the level of the SCI may be experienced
Nausea and bradycardia may occur
Patients may also be extremely anxious and can have a feeling of impending doom

*Adapted from Consortium for Spinal Cord Medicine (1997)

of the stimulus) up the spinothalamic and posterior columns, until they are blocked at the level of the lesion and prevented from reaching the cerebral cortex. The major splanchnic sympathetic outflow at level T5–L2 is stimulated by the impulses, resulting in vasospasm and hypertension. The patient experiences a severe headache, as his or her blood pressure rapidly rises. The parasympathetic nervous system is unable to counteract the sympathetic outflow, due to the SCI, and hypertension cannot therefore be adequately regulated (Porth, 1998; Travers, 1999).

Hypertension is detected by baroreceptors in the aortic arch, carotid sinus, and vasomotor centre of the medulla. The vasomotor centre responds by sending parasympathetic impulses, via the vagus nerve inducing bradycardia, and vasodilatation above the level of injury, in an attempt to lower the blood pressure. The resulting vasodilatation results in the individual becoming flushed in the face and sweaty. Less commonly, some patients also develop nasal passage congestion (Hanak and Scott, 1993; Clinchot and Colachis, 1996; Grundy and Swain, 1996; LaFavor and Ang, 1997; Pasquina *et al*., 1998).

Since the parasympathetic impulses cannot descend the cord past the lesion, their effects are only manifested above this level. The sympathetic vasoconstrictive response will therefore continue uninhibited and will continue to elevate the blood pressure. Hypertension may become as high as 200–300 mmHg systolic (Cormarr and Eltorai, 1995). If left untreated, it may result in convulsions, cerebral haemorrhage, cardiac arrhythmia, and eventually death (Pine *et al*., 1991; Clinchot and Colachis, 1996; LaFavor and Ang, 1997; Pasquina *et al*., 1998).

NURSING CARE OF THE SCI PATIENT

The major nursing objectives when caring for people with spinal cord lesions are listed in Table 2.

In the ICU, a holistic approach is essential in order to prevent the various problems presented by an SCI patient. However, some factors are particularly important to consider in the context of autonomic dysreflexia. One of the main problems is that a patient with an SCI may not experience the discomfort and pain that would warn a 'normal' patient of noxious stimuli, due to loss of sensation below the level of the SCI and a restricted visual field (Partridge, 1994). This situation may be further compromised by a reduced ability to communicate, e.g. due to sedation, intubation or cerebral trauma. Regular changes of position and skin inspections are required to relieve pressure and prevent painful stimuli. To maintain optimal skin condition, a dietician should advise on nutritional support.

Bladder and bowel management is very important for paralysed people. Distension of the bowel and bladder may precipitate autonomic dysreflexia. Inserting catheters using strict sterile techniques helps to prevent urinary tract infections. The use of enough lubricant when manually evacuating bowels – with only one finger – is essential to prevent over-stimulation and the onset of autonomic dysreflexia (Wood and Grundy, 1996).

Table 2. Major nursing objectives of caring for people with spinal cord lesions*

Nursing objectives
▸ Identify problems and prevent deterioration
▸ Prevent secondary complications
▸ Facilitate maximum functional recovery for each individual
▸ Support patients and their families, as they adjust to the patient's changed physical status
▸ Be aware of the effect of the injuries on the patients' self-esteem, giving high priority to establishing a new sense of self-worth
▸ Educate patients and their relatives, in all aspects of care needed to maintain their well-being

*Adapted from Wood and Grundy (1996)

PRACTICE GUIDELINES FOR AUTONOMIC DYSREFLEXIA

Serious consequences of undiagnosed and unmanaged autonomic dysreflexia include retinal haemorrhage, apnoea, cerebrovascular accident, renal failure, subarachnoid haemorrhage, seizures, cardiac dysrhythmias, cardiac arrest and death. Effective nursing interventions may well prevent autonomic dysreflexia.

The Consortium for Spinal Cord Medicine (1997), supported by the Paralyzed Veterans of America, produced a clinical guideline for the management of autonomic dysreflexia, which is based on scientific evidence, expert consensus and clinical input (Bowers, 1998). It gives recommendations for the care of individuals with a SCI at or above T6, who present with an acute onset of signs and symptoms of autonomic dysreflexia, and provides evidence-based reasoning for each action. This guideline is presented below.

ASSESSMENT OF PATIENTS WITH SCI

All patients with a SCI should be assessed for risk factors that predispose them to the development of autonomic dysreflexia (Table 3).

Once the risk has been identified, all providers of health care should be alerted and provided with information regarding the causes and management of autonomic dysreflexia (Dunn, 1991). Assessment of the individual with signs and symptoms of autonomic dysreflexia should include looking for the presence of precipitating factors. Nurses need to be aware that any stimulus which could cause pain or discomfort in an able-bodied individual can result in autonomic dysreflexia in a paralysed patient.

PREVENTION OF AUTONOMIC DYSRFLEXIA

Autonomic dysreflexia can often be prevented by the use of good nursing practices and a knowledge of precipitating factors. Individuals with SCI and all their caregivers should be taught these practices and their skills and knowledge evaluated (LaFavor and Ang, 1997).

Interventions aimed at preventing episodes of autonomic dysreflexia are noted in Table 4 and continuing assessment of the SCI patient should consider many factors (Table 5).

Table 3. Risk factors associated with autonomic dysreflexia

Risk factors
▸ Level of spinal cord injury at T6 or above but may occur with injuries as low as T10 (Cormarr and Eltorai, 1995)
▸ Any recent episode of autonomic dysreflexia and known causative stimulus, e.g. bladder or bowel problems
▸ Episodes of headache, which could be due to hypertension due to autonomic dysreflexia
▸ Blood pressure elevation >20 mmHg above baseline, indicating mild hypertension which may lead to autonomic dysreflexia
▸ Altered bladder or bowel function and management – the most common causes of noxious stimuli leading to autonomic dysreflexia
▸ Altered skin integrity, which could provide a source of noxious stimuli
▸ Pain or pressure, which could provide a source of noxious stimuli
▸ Some drugs may cause bladder contraction (Vaidyanathan and Krishnan, 1996)

Table 4. Preventative interventions for autonomic dysreflexia

Interventions
▸ Monitoring urinary output and making necessary changes to the bladder management programme to prevent over-distension
▸ Assessing for urinary tract infection, acquiring appropriate treatment and evaluating effectiveness
▸ Use of an anaesthetic jelly for catheterisation and rectal examination
▸ Monitoring the bowel management programme and making necessary changes to the programme to prevent constipation or impaction (Dunn, 1991; Huston and Boelman, 1995)
▸ Providing appropriate skin and wound care to prevent noxious stimuli, e.g. from pressure sores, skin infections, ingrown toenails, tight clothing, wrinkled sheets (Hall and Young, 1983; LaFavor and Ang, 1997)
▸ Providing other treatment measures, as appropriate, to alleviate the cause of noxious stimuli (see the causative factors listed in Table 5)
▸ When planning invasive diagnostic procedures, especially of the genitourinary and gastrointestinal system [the most common causes of autonomic dysreflexia from stimulation (Grundy and Swain, 1996; LaFavor and Ang, 1997; Pasquina *et al.*, 1998)] collaborate with medical colleagues regarding prophylactic administration of antihypertensive drugs
▸ Use of painkillers and anaesthetic gel or ointments for painful procedures below the level of injury to the spinal cord

Table 5. Continuous assessment of the spinally injured patient*

Bladder/genitourinary system	Skin
▸ Bladder over-distension	▸ Pressure ulcers/treatment
▸ Urinary tract infection	▸ Ingrown toenails
▸ Bladder or kidney stones	▸ Burns or sunburn
▸ Invasive testing	▸ Blisters
▸ Urinary sphincter spasms	▸ Insect bites
▸ Scrotal compression/epididymitis	▸ Contact with hard or sharp objects
Bowel/gastrointestinal system	Reproductive system
▸ Bowel distension	▸ Menstruation
▸ Impaction	▸ Pregnancy, especially labour and delivery
▸ Gallstones	▸ Vaginitis
▸ Gastric ulcers/gastritis	▸ Sexual intercourse
▸ Invasive testing	▸ Ejaculation
▸ Haemorrhoids	
▸ Gastrocolic irritation	Other precipitating factors
▸ Appendicitis	▸ Constrictive clothing, shoes or appliances
▸ Abdominal pathology or trauma	▸ Heterotopic bone
	▸ Fractures or trauma
Cardiovascular system	▸ Surgical or diagnostic procedures
▸ Deep vein thrombosis	▸ Pain
▸ Pulmonary oedema	▸ Temperature fluctuations

*Adapted from LaFavor and Ang (1997)

MANAGEMENT PRINCIPLES OF AUTONOMIC DYSREFLEXIA

The principles for effective management of autonomic dysreflexia are described below (after Consortium for Spinal Cord Medicine, 1997) and are summarised in Table 6.

Blood pressure

If autonomic dysreflexia is suspected, check the patient's blood pressure. Elevated blood pressure can be life-threatening and may need immediate investigation and treatment. If the blood pressure is not elevated, refer the patient to a medical colleague for diagnostic assessment, as other medical problems may cause similar signs and symptoms.

If the blood pressure is elevated, sit the patient up immediately (or raise the head of the bed if the patient's clinical condition does not allow the patient to be sat up). This promotes pooling of blood in the lower extremities and may reduce blood pressure. Loosen any clothing or restrictive devices, which may allow a pooling of blood in the abdomen and lower extremities. Monitor the blood pressure and heart rate frequently (every 2–5 minutes until stable). The blood pressure may fluctuate during an autonomic dysreflexia episode.

Table 6. Treatment of autonomic dysreflexia

Treatment intervention
▸ The patient should be assisted to a sitting position or the head of bed elevated to a 90°angle. This helps to lower blood pressure by using postural drainage
▸ Any constrictive clothing or equipment, such as abdominal binders, belts, clothes, or anti-embolic stockings, must be loosened or removed. This helps to reduce impedance to venous return and/or may relieve the cause of noxious stimuli
▸ Blood pressure and pulse should be monitored every 2–3 minutes throughout the episode of autonomic dysreflexia. This enables the patient to be monitored closely for excessive hypertension and assesses the effectiveness of any drug treatment given

Find the cause

The possible causes of autonomic dysreflexia listed in Table 5 should be systematically assessed until the stimulus is determined and eliminated. This management of autonomic dysreflexia is endorsed by other authorities (Hanak and Scott, 1993; Huston and Boelman, 1995; Clinchot and Colachis, 1996; LaFavor and Ang, 1997; Pasquina *et al.*, 1998).

Quickly survey the patient for possible causes of autonomic dysreflexia, beginning with the urinary system.

> *It is critical to identify the cause of autonomic dysreflexia as soon as possible. Removal of the stimuli usually causes the symptoms to abate*
>
> (Grundy and Swain, 1996; LaFavor and Ang, 1997; Pasquina *et al.*, 1998).

Bladder distension: The most common cause of autonomic dysreflexia is bladder distension. If an indwelling urinary catheter is not in place, a catheter should be inserted. Prior to inserting the catheter, instil lignocaine jelly into the urethra and wait several minutes. Catheterisation can exacerbate autonomic dysreflexia. The lignocaine jelly may decrease the sensory input and relax the sphincter to facilitate catheterisation.

If the person has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions or obstructions. Make sure that the catheter is correctly inserted. If a problem is found, correct it immediately.

If the catheter appears to be blocked, gently irrigate the bladder with a small amount of fluid, such as normal saline at body temperature. Avoid manually compressing or tapping on the bladder, because this may increase sensory input and exacerbate autonomic dysreflexia. Similarly, use of a larger volume of fluid or of a cold solution might irritate the bladder and exacerbate autonomic dysreflexia.

If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter at once. Irrigating and changing the catheter should be done as quickly as possible.

Pharmacological management may become necessary if the blood pressure remains elevated and/or if catheter replacement is difficult. Monitor the patient's blood pressure during bladder drainage. Sudden decompression of a large volume of urine might produce hypotension.

Treat severe (symptomatic) hypotension by laying down the individual and elevating the legs. Additional corrective measures are not usually required. However, if indicated intravenous fluids and adrenergic agonists should be given.

If the catheter is draining and the blood pressure remains elevated, suspect faecal impaction.

Faecal impaction: The opinions reviewed by the Consortium for Spinal Cord Medicine (1997) differed on whether or not it was more important to control hypertension before digital stimulation to treat faecal impaction (which may further exacerbate autonomic dysreflexia). The Consortium recommends giving an antihypertensive agent with rapid onset and short duration to treat the hypertension while the causes of autonomic dysreflexia are being investigated. Nifedipine and nitrates are the most commonly used agents.

Faecal impaction is the second most common cause of autonomic dysreflexia.

If faecal impaction is suspected, check the rectum for stool, using the following procedure. With a gloved hand, instil a topical anaesthetic agent, such as lignocaine jelly generously into the rectum. Wait approximately 5 minutes for sensation in the area to decrease. Then, with a gloved hand, insert a lubricated finger into the rectum and check for the presence of stool. If present, gently remove, if possible. If autonomic dysreflexia becomes worse, stop the manual evacuation immediately. Instil additional topical anaesthetic and re-check the rectum for the presence of stool after approximately 20 minutes. A rectal examination may exacerbate autonomic dysreflexia. Instillation of a local anaesthetic agent may decrease the occurrence of autonomic dysreflexia.

PATIENT MONITORING AFTER EPISODE RESOLUTION

Monitor the patient's blood pressure and symptoms for at least 2 hours after resolution of the autonomic dysreflexia episode to make sure that it does not recur. The hypertension and symptoms may have resolved because of the medication rather than treatment of the cause. Symptoms managed by pharmacological treatment may begin to reverse themselves within this timeframe.

DRUG TREATMENT

If there is a poor response to the treatment specified above and/or the cause of the autonomic dysreflexia has not been identified, pharmacological control of the blood pressure will be required while the causes of autonomic dysreflexia are further investigated.

Pharmacological treatment of autonomic dysreflexia aims to avert or abate a hypertensive crisis using vasodilator agents, alpha-blocking agents, ganglionic-blocking agents, or calcium-channel blocking agents. These agents decrease sympathetic adrenergic and cholinergic outflow or block adrenergic receptors (Clinchot and Colachis, 1996). Although many drugs have been reported to be effective in acute and recurrent episodes of autonomic dysreflexia, there is little experimental evidence available to support these claims (Pasquina *et al*., 1998).

EVALUATING TREATMENT OUTCOME

The effectiveness of treatment may be evaluated according to the following outcomes:

- The cause of the autonomic dysreflexia episode has been identified.
- The blood pressure has been restored to normal limits for the patient (usually 90–110 mmHg systolic for a tetraplegic person in the sitting position).
- The heart rate has been restored to normal limits and the patient is comfortable, with no signs or symptoms of autonomic dysreflexia, increased intracranial pressure, or heart failure.

DOCUMENTATION OF EPISODE

The episode of autonomic dysreflexia should be clearly documented in the patient's medical record. The presenting signs and symptoms, their course, the treatment given, recordings of blood pressure and heart rate, and the response to treatment should all be noted.

MANAGEMENT REVIEW

When the episode of autonomic dysreflexia has been stabilised, the precipitating cause should be reviewed with the patient and his family or caregivers. This process entails adjusting the treatment plan to ensure that future episodes are recognised and treated, or avoided altogether. The process also includes a discussion of autonomic dysreflexia in the SCI patient's education programme, so that he or she will be able to recognise its early onset and obtain help as quickly as possible.

A person with SCI should be given a written description of treatment for autonomic dysreflexia at the time of discharge that can be referred to in an emergency (Consortium for Spinal Cord Medicine, 1997).

SUMMARY

Current theories suggest that autonomic dysreflexia is the result of a massive discharge that occurs uninhibited in patients with spinal cord lesions above the major sympathetic splanchnic outflow at T6. This sympathetic discharge is thought to occur as a reflex phenomenon.

Autonomic dysreflexia is a potentially life-threatening syndrome associated with the elevation of blood pressure (Pine *et al*., 1991; Clinchot and Colachis, 1996; LaFavor and Ang, 1997; Pasquina *et al*., 1998). All SCI patients, in all settings, at any time, are at risk of developing autonomic dysreflexia.

The timely assessment and planning of care and management are essential for all nurses caring for patients at risk of developing autonomic dysreflexia (LaFavor and Ang, 1997). By referring to the guidelines outlined in this paper, ICU nurses have the potential to improve the quality and consistency of their care of paralysed patients.

It is essential that the signs and symptoms of autonomic dysreflexia are recognised early, so allowing time for appropriate and effective intervention to be undertaken to prevent a hypertensive crisis. If blood pressure does become highly elevated, emergency intervention is necessary, which may include the use of drug therapy.

Clinically effective care and improved morbidity for SCI patients are the intended outcomes. Adopting a systematic process when autonomic dysreflexia occurs will enhance clinical decision-making. However, prevention is better than cure, and good nursing care is of the essence.

Furthermore, since the patient with SCI is treated by a multidisciplinary team, the whole team should be educated about the signs, symptoms and management of autonomic dysreflexia (Adsit and Bishop, 1995).

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